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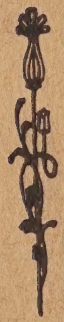
THE MANAGEMENT OF CRANIO-CEREBRAL INJURIES



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The Management of Cranio-Cerebral Injuries*

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WHEN an individual receives an injury to the head it is well to be cautious regarding the prognosis. Even though the patient appears normal, haemorrhage from the middle meningeal artery may be occurring and the patient will become dull in a few hours, and unconscious shortly afterwards. Or a patient with a severe laceration of the brain may seem well for forty-eight hours and then die in the next twelve. Several months after an injury all the symptoms of increased intra-cranial pressure may appear as the result of a venous haemorrhage on the surface of the brain, or later still, the liquified and encysted blood clot may cause epilepsy, mental deterioration, headache, etc. Most often, of course, the individual who appears normal shortly after an injury, remains well. Head injuries are now becoming so common that it behooves all of us to have a fairly satisfactory working knowledge of these cases.

The patient is often badly shocked when first seen. He will be white, cold, have a low blood-pressure, a fast pulse, and the breathing will be shallow and rapid. The first consideration must be the treatment of this shock, and this treatment may be carried out in the following way:

The patient is put between blankets with plenty of hot-water bottles; the foot of he bed is raised, hot coffee is given by rectum. The extremities are bandaged to support the peripheral circulation. Pituitrin and caffein may be given, but not morphia. Patients with cerebral injuries stand morphia very badly, especially if there is any tendency to respiratory failure. Furthermore, the morphia may mask subsequent important signs.

A chart of the respiration, pulse and blood-pressure should be commenced immediately, and charted every half-hour. If feasible a special nurse is obtained, also at this time certain general observations may be made which will not disturb the patient.

(a) Note if there is any obvious injury to the scalp or bleeding from the ears or nose.
(b) The patient's response to questions.
(c) Any obvious loss of movement in the limbs.
(d) The comparative size of the pupils.

*A lecture delivered to the fifth year students at the University of Toronto.

After the Shock Has Passed Off.

One should deal immediately with a laceration of the scalp and a possible underlying comminuted fracture. The area is widely shaved. Anaesthesia is obtained by surrounding the area with an injection of 1% novocaine between the scalp and bone. If the edges of the laceration are crushed and soiled they are cleanly incised. Bleeding is controlled by turning back the aponeurosis of the scalp with a row of fine haemostats. No attempt is made to catch individual bleeding points. The underlying bone is carefully palpated and examined for evidence of fracture. If the fracture is a simple fissure, the wound in the scalp should be carefully sutured, by placing fine silk interrupted sutures sufficiently close to control haemorrhage. No drainage is necessary. If there is a depressed comminuted fracture, loose fragments of bone must be removed, as they are very likely to become infected. There is a certain urgency in dealing with these cases, as any infection may bring about a meningitis or brain abscess. If the dura is intact, under no consideration incise it in the presence of this infected field (fear of meningitis); if the dura has been torn, and the area is soiled, remove by suction and gentle swabbing any debris and obviously severely traumatized brain tissue. Only a small opening should be left through the dura, otherwise a large herniation of the brain may result. A cigarette drain of rubber tissue is left down to this area, and this should be removed in forty-eight hours, if there is no obvious evidence of infection. Any bone defect can be repaired at a later date if necessary.

The neurological examination is completed as soon as possible. If the patient has regained consciousness and is co-operating well, all the cranial nerves should be examined. The arms and legs should be tested for strength, changes of sensation, and reflexes. This record may be most valuable in determining treatment in the subsequent few hours or days. Many of these patients prove later to be medico-legal cases and one may be chagrined in court if one's record is not complete. If the patient remains unconscious, this record is necessarily curtailed. The chart of the respirations, pulse, and blood-pressure is carried on. A lumbar puncture is done and the pressure estimated. This pressure can only be recorded satisfactorily with a manometer; the rate at which the fluid flows through the needle is very fallacious. The normal pressure with a mercury manometer is from 7 to 12 mm. With the Ayer manometer, the pressure is recorded by the height of the column of cerebral-spinal fluid and is from 10 to 15 cms. If there is blood in the C.S.F., several ccs. are collected in three test tubes, and if there is as much blood in the third test tube as in the first, one may assume that the bleeding was not caused by the puncture.

Stereoscopic plates of the skull, taken from both sides and from the front and back, are made when the patient can be safely moved. If the problem of diagnosis of compression from a torn middle meningeal has to

be met, it is a great help if it is known whether a linear fracture is present which might have torn the vessel.

There may be bleeding or a cerebro-spinal fluid leak from the auditory canal (fracture of the base, through the petrous bone). If so, the external canal should be thoroughly cleansed with one in a thousand bichloride solution and a pledget of cotton soaked in this solution placed lightly in the canal, and changed every hour. Every attempt must be made to keep this area clean, as infection is very likely to set up a meningitis. The ear should not be syringed.

Having accumulated as much information as possible from the chart recording the respirations, pulse, blood-pressure, and cerebro-spinal fluid pressure, and having made as complete a neurological examination as possible, one is now in a position to consider whether the patient is suffering from concussion only, or whether he is showing signs of compression, or failure of the medullary centres.

Concussion.

This term may be very confusing, and it is not always clear just what is meant. It may be considered this way: if an individual receives a blow, he may be only dazed, and if one could see the brain, there would probably be no apparent microscopic changes. A more severe injury might bring about loss of consciousness, and examination of the brain might show small microscopical haemorrhages scattered through it, and even gross lacerations. By concussion then we mean injury to the brain, which may only daze an individual, or render him unconscious. The injury might not be apparent even with a microscope, on the contrary there might be a gross laceration.

With this conception of concussion, the clinical symptoms vary according to the degree, and situation of the injury to the brain. Usually there is a period of unconsciousness which may be of short duration, or extend over many hours. Following this there is the stage of reaction frequently ushered in by vomiting and attended by various symptoms described at some length in the text books. The variation in the symptoms may be explained by the degree of interference with the physiological functions of the different parts of the brain; for instance, prolonged irritability from injury to the frontal lobes, convulsions from cortical irritation, paralysis or sensory changes from injury in the Rolandic area, vertigo from injury to the internal ear or cerebellar path-ways. Vomiting and abnormalities in the pulse, respirations or temperature from injury to the medulla.

Treatment of Concussion.

In addition to general symptomatic treatment and the avoidance of morphia, there should be absolute quiet and prolonged rest in bed. Luminol in two grain doses is a satisfactory sedative. As much as ten or twelve

grains may be administered in twenty-four hours, usually by mouth, but it may be given hypodermically.

Compression.

By compression we mean that the brain is being compressed either by oedema or haemorrhage. There are certain signs which enable us to determine when this compression is taking place, and it is most important to be able to appreciate this and to institute proper treatment.

Compression due to extra-dural haemorrhage is almost always brought about by a tear of the middle meningeal artery. The usual syndrome of such a pathological condition is as follows. It is quite possible that the patient has had a fairly lucid interval after his initial injury, he will then commence to show the first and most important sign of compression, namely, dullness and stupour which may go on to complete loss of consciousness. Loss of consciousness from concussion may be so prolonged that there is no lucid interval, and one then has to depend upon the other signs of compression:

1. The breathing becomes loud and stertorous and may drop down to four or five a minute. The patient can be heard breathing in the next room. Expiration and inspiration are about equal.

2. The blood-pressure rises.

3. The pulse becomes slowed and fuller.

4. The cerebro-spinal fluid pressure mounts.

(Hence the importance of keeping a chart of these readings right from the start, so that one may know beyond a doubt that these changes are taking place).

Having detrmined that compression of the brain is taking place, one makes sure that a middle meningeal vessel is not bleeding, and this involves a split vertical temporal muscle incision about a quarter of an inch in front of the ear, two inches long, with the lower end at the zygomatic bone. If there is no bleeding beneath the temporal muscle, it is not likely that there has been a linear fracture which would bring about a tear of the vessel; however, it is not well to depend entirely upon this sign, as it takes only another few minutes to take out a button of bone to see if there is an underlying clot. If there is a clot, the dura is not opened. The bone need not be replaced, the temporal muscle and scalp are quickly closed and the same procedure is carried out on the other side. A local anaesthesia with 1% novocaine may be used, or if the patient is deeply unconscious, no anaesthetic is required. If an extra-dural clot is found, the temporal muscle is retracted and the bone opening enlarged to two inches in diameter with rongeurs. The clot is then removed by suction and swabbing. With a good light, one is usually able

to see the bleeding point, and can carefully pass a ligature around the vessel in the dura, taking care not to wound an underlying vessel in the brain. If a Cushing silver clip is available, it is much easier to use this in place of the ligature. However, if the bleeding point cannot be clearly seen, light packing with gauze will control the bleeding sufficiently, until one can get better facilities with which to work; even if the bleeding point is not stopped, the compression is relieved with the large opening in the bone. These patients die from compression, not loss of blood.

There are certain lateralizing signs which are of value in operating on a suspected middle meningeal haemorrhage:

1. The pupil on the side of the lesion may be large, due to paralysis of the third nerve, brought about by more or less direct pressure of the clot.

2. A linear fracture seen in the X-ray is a guide to the side.

3. There may be some weakness of the arm and leg on the opposite side.

It is important to operate on the other side, if nothing is found on the side first explored. Furthermore, remember that concussion with unconsciousness may be prolonged so that there is no lucid interval. In such a situation an exploratory operation is justified when there is evidence of compression.

If the compression is not due to extra-dural haemorrhage, it is likely brought about by a general oedema of the brain resulting from traumatism, and this may be treated in the following manner:

1. The return flow of blood from the brain is greatly facilitated by the respiratory movements of the chest. How disastrous to one of these unconscious patients to have him lying on his back with the respirations largely obstructed by the tongue. There is no more efficient method of increasing oedema of the brain and hastening death. The patient should be placed on his side and the tongue kept well forward, the head is elevated. It may be necessary to place tubes in the nares and inside the cheek to obtain a free airway.

2. Sufficient cerebro-spinal fluid is slowly withdrawn by lumbar puncture every two hours to reduce the pressure to half, that is, if the pressure is found to be thirty cms., it should be reduced to fifteen. The amount removed in these punctures must be determined by manometer readings, as it is possible to remove too much fluid and so bring about herniation of the medulla and cerebellum into the foramen magnum.

3. Two ounces of concentrated sulphate solution may be left in the rectum every two hours, or if not retained, it is usually easy to introduce it into the stomach with a nasal catheter.

4. Concentrated solutions may be used intra-venously, probably the best is 75 cc. of 50% glucose. This hypertonic solution in the blood-stream brings about a flow of fluid from the brain to the blood-stream and so reduces the oedema. Glucose is better than sodium chloride, which in the past has been commonly used in a 15% solution. Sodium chloride solution quickly brings about a reduction in the oedema of the brain, but later the salt gets into the tissues and brings about a flow the other way. The glucose, on the other hand (taken up by the liver and muscles), does not find its way into the brain and bring about this reverse flow. The concentrated glucose solution is conveniently obtained in 20 cc. ampules.

By these two forms of treatment, one surgical and the other non-surgical, many patients suffering from compression may be saved by preventing failure of the medullary centres.

Failure of the Medullary Centres.

The medullary centres have been responding, but they may commence to fail, and the picture is now changed. The pulse becomes rapid and irregular, the blood-pressure drops, the breathing, although still loud, becomes very rapid. This is the beginning of the end, and one can say almost with certainty that the patient will die.

The Simple Depressed Fracture.

There is no urgency about the treatment of this condition, as there is not the fear of infection that there is with a compound fracture. The fracture, however, should be re-elevated within three or four days. The procedure is usually carried out quite simply by turning down a flap of scalp and re-elevating the fragments piecemeal. Even if the fragments are detached from all blood supply they are left (note difference in compound fractures). It has been found from clinical experience that if these simple depressed fractures are not elevated the patient is likely to suffer subsequently from headache, epilepsy, or even have signs of pressure.

Summary.

In the management of cranio-cerebral injuries the important points are:

1. To treat the shock.

2. To record carefully from the start the necessary observations.

3. A laceration of the scalp is carefully dealt with.

4. A compound depressed fracture should be immediately and specifically dealt with after recovery from the shock.

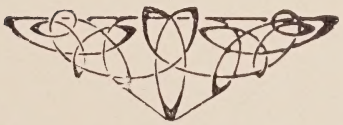
5. Observation is maintained for the signs and symptoms of compression. Compression is usually due to (a) extra-dural haemorrhage (to be treated surgically), or oedema of the brain (see treatment).

6. Simple depressed fractures demand operation, but operation is not so immediately urgent as in the case of compound fractures.

7. Be guarded about the prognosis for at least three days even after seemingly trivial cases of concussion.

NOTE 1.—There is considerable controversy regarding the advisability of surgical intervention in cases of injuries to the brain giving definite localizing signs, and thought to be due to subdural haemorrhage, laceration or intra-cerebral haemorrhage. Only very exceptionally is it wise to operate on these cases. Sub-temporal decompression has been advocated in cases of compression thought to be due to oedema, and which cannot be controlled by the measures outlined. The turning down of large parieto-occipital bone flaps with the opening of the underlying dura and the replacing of the flaps loosely in position, has also been considered. Cerebellar decompression has also been suggested. It is certainly unwise in almost all instances to attempt any intradural surgical procedure when dealing with these cases.

NOTE 2.—The clean-cut, classical syndromes of concussion compression and medullary failure are more than often not seen. One syndrome may follow the other so quickly that the picture is blurred. If the signs and symptoms of medullary failure follow quickly on those of shock and compression, very little can be done for the patient.



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